Sexually Transmitted Parasitic Diseases

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ABSTRACT

An increasing number of diseases are recognized as being sexually transmitted. The majority of these are bacterial or viral in nature; however, several protozoan and nematode infections can also be transmitted by sexual activity. For most of these diseases, the primary mode of transmission is nonsexual in nature, but sexual activity that results in fecal-oral contact can lead to transmission of these agents. Two parasitic diseases commonly transmitted by sexual contact are amebiasis and giardiasis. The management of these conditions is discussed.

KEYWORDS: Amebiasis, giardiasis, parasites

Objectives: Upon completion of this article, the reader should be familiar with and understand the management of amebiasis and giardiasis.

An increasing number of diseases are recognized as being sexually transmitted. The majority of these are bacterial or viral in nature; however, several protozoan and nematode infections can also be transmitted by sexual activity. For most of these diseases, the primary mode of transmission is nonsexual in nature, but sexual activity that results in fecal-oral contact can lead to transmission of these agents. Two parasitic diseases commonly transmitted by sexual contact are amebiasis and giardiasis.

AMEBIASIS

Etiology

Amebiasis is caused by the protozoan *Entamoeba histolytica*. It exists in two forms, the trophozoite and the cysts. The trophozoite is motile by means of pseudopodia. It multiplies by binary fission within the intestinal lumen. Unless diarrhea is present, trophozoites become rounded and develop into cysts within the intestinal lumen before being excreted into the stool.[1] The trophozoite is very fragile and cannot encyst after excretion outside the body. Mature cysts are quadrinucleate and $\sim$12 μm in diameter. When ingested by a susceptible host, the wall of the cyst degenerates in the small intestine, releasing a single amoeba. This single amoeba then divides into eight uninucleated amoebas. They then migrate to the large intestine and develop into trophozoiotes, which then encyst and complete the life cycle.[1]

Disease Pathogenesis

Infection with *E. histolytica* may be asymptomatic, or it may result in dysentery or extraintestinal disease. In most infections the trophozoites aggregate in the intestinal mucin layer and form new cysts, resulting in a self-limited and often asymptomatic infection. Colitis results when the trophozoite penetrates the intestinal mucosal layer.[2] Invasion is mediated by the killing of epithelial cells, neutrophils, and lymphocytes by the trophozoite. Neutrophils responding to this invasion contribute to

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cellular damage at the site of invasion. Once the intestinal epithelium is invaded, extraintestinal spread to the peritoneum, liver, and other sites may occur.[3]

Amebiasis is transmitted primarily by the fecal-oral route, most commonly from contaminated drinking water or by unsanitary food handling. *E. histolytica* is often found in the stool of homosexual men and is the most common intestinal parasite seen in gay communities throughout the world. Sexual behavior such as analingus or fellatio after anal-genital intercourse can lead to infection. Direct rectal inoculation of trophozoites can occur but is uncommon.[4]

Clinical Presentation

COLITIS

The incubation period from ingestion of cysts to the development of symptoms ranges from days to months, but averages 2 to 4 weeks.[1] Many patients with *E. histolytica* are asymptomatic.[3] Gastrointestinal symptoms may be mild or severe in intensity. Patients with amoebic colitis typically present with a several-week history of crampy abdominal pain, weight loss, and watery or bloody diarrhea. The differential diagnosis includes other infectious causes of diarrhea (*Salmonella, Shigella, Campylobacter,* and enteroinvasive *E. coli* infection) as well as noninfectious causes of diarrhea such as inflammatory bowel disease and ischemic colitis.[3] Unusual manifestations of amoebic colitis include necrotizing colitis and toxic megacolon.[5] An amoeboma, a mass of concentric granulation tissue, may form. This is most frequently seen in the cecum or ascending colon and may mimic colon carcinoma.

AMOEBIC LIVER ABSCESS

Patients with amoebic liver abscesses typically develop symptoms fairly quickly, over a period of 2 to 4 weeks. Symptoms include fever, right upper quadrant abdominal pain, and cough. Involvement of the diaphragmatic surface of the liver can result in pleuritic-type chest pain or referred shoulder pain. Symptoms of amoebic colitis may also be present. Complications can result if the abscess ruptures into the peritoneal cavity, the pleura, or the pericardium. The differential diagnosis of an amoebic abscess includes pyogenic liver abscess, eichonoccal cyst, and necrotic tumor, such as hepatoma.[6,7]

Diagnosis

In developing countries, amebiasis is usually diagnosed by the detection of motile trophozoites or cysts on a saline wet mount from a stool specimen. This is associated with a low sensitivity and high false-positive rate due to the finding of nonpathogenic parasites in the stool.[2] Several alternative tests are used to diagnose intestinal amebiasis (Table 1). The diagnosis is frequently made by the detection of *E. histolytica*-specific antigens or DNA in the stool and by the presence of antiamebic antibodies in the serum. The antibody titer may remain positive for years.[8,9]

Table 1 Sensitivity of Tests for the Diagnosis of Intestinal and Extraintestinal Amebiasis*

Test	Colitis	Liver Abscess
Microscopy		
Stool	25–60%	10–40%
Abscess fluid	NA	<20
Antigen detection		
Stool	90%	40%
Serum	65% (early)	100% (before treatment)
Abscess fluid	NA	40%
Indirect hemagglutination (Ab)		
Acute serum	70%	70–80%
Convalescent serum	$>90\%$	$>90\%$

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Endoscopic biopsies generally show a range of nonspecific inflammatory changes in the colonic mucosa. The finding of amoeba in the mucus exudate is rare, but can be aided by staining with periodic acid-Schiff, or immunoperoxidase and antilectin stains.[10]

Treatment

Treatment is recommended, even for asymptomatic carriers, to eliminate the risk of future transmission. Therapy for invasive infection differs from that for noninvasive infection. Noninvasive infection can be treated with paromomycin. Metronidazole is the mainstay of the treatment for invasive infection. Approximately 90% of patients with mild to moderate amebic dysentery have a response to treatment with metronidazole. Parasites may persist in the stool in as many as 40 to 60% of patients after treatment with metronidazole, and therefore treatment should be followed with paromomycin. In the rare case of fulminant amebic colitis, the addition of broad-spectrum antibiotics to cover enteric gram-negative rods is often necessary. Surgery is sometimes required for fulminant colitis or toxic megacolon.[3]

GIARDIASIS

Etiology

Giardiasis is caused by infection with the protozoan *Giardia lamblia.* It occurs in two forms, a motile trophozoite and an environmentally stable cyst. The trophozoites are teardrop shaped and contain two

distinct nuclei, four pairs of flagella, and an adhesive disc on their concave ventral surface. The mature cysts are oval and contain four nuclei.[11]

The life cycle of *Giardia lamblia* begins with ingestion of mature cysts. The trophozoites are released in the duodenum. Using its adhesive disc, the trophozoites attach themselves to the epithelial brush border of the duodenum and jejunum. The attachment is substantial and results in disc impression prints when the organism detaches from the surface of the epithelium. The trophozoites then feed on the mucous secretions of the gastrointestinal tract, but do not penetrate the mucosa. The trophozoites divide by means of longitudinal binary fission, producing two daughter trophozoites. The epithelial surface sloughs off the tip of the villous every 3 days. The cysts detach, enter the fecal stream, and encyst as the trophozoites pass throughout the colon. Both cysts and trophozoites may be excreted in the stool. The trophozoites are fragile and rapidly degenerate outside of the host, but the cysts are very stable and may survive for several months.

Transmission of *Giardia lamblia* occurs exclusively by the fecal-oral route. Consumption of contaminated water or sexual activity with an infected individual are the most common causes of infection. The disease is most common in subtropical regions. In the United States, a prevalence of 1 to 2% is found among urban inhabitants. Epidemics of giardiasis are common in industrialized countries and can usually be traced to a contaminated water source. It is increasingly recognized that the prevalence among populations of homosexual men is higher than in heterosexual controls.[12] Prevalence rates of 4 to 18% have been described in both North American and European male homosexual populations.[11,13]

Clinical Presentation

The incubation period of *Giardia lamblia* is about 2 to 3 weeks. The acute phase of *Giardia* infection begins shortly thereafter and lasts for a few days. Onset may be accompanied by nausea, anorexia, malaise, and low-grade fever. Diarrhea occurs in up to 80% of patients. The stools are loose, characteristically foul smelling, contain fat and mucus, and are often described as floating in the toilet bowl. Patients may complain of crampy right upper quadrant abdominal pain or epigastric abdominal pain. Proctitis due to *Giardia lamblia,* is usually only seen in male homosexuals. Occasionally the gallbladder is involved and may produce symptoms of biliary colic and jaundice. Importantly, up to 20% of patients may be asymptomatic.[14]

The acute phase may be followed by a subacute or chronic phase. Symptoms in these patients include recurrent, brief episodes of loose, foul-smelling stools. This may be associated with abdominal distention and increased flatulence.

Diagnosis

The differential diagnosis of acute giardiasis includes other causes of acute diarrheal illness, including viral or bacterial gastroenteritis, food poisoning, and amebiasis. Inflammatory bowel disease, malabsorption due to pancreatic insufficiency, or celiac sprue must be considered in patients with chronic symptoms. In homosexual men with proctitis, infection with *Entamoeba histolytica, Neisseria gonorrhea, Chlamydia trachomatis,* or herpes simplex should be considered.

Routine stool examination is usually recommended to identify intestinal parasites. However, because *Giardia lamblia* attaches so securely to the mucosal surface, a series of five or six stool specimens may still fail to identify the organism. In addition, cysts are often excreted in a cyclic fashion. However, the diagnosis of giardiasis is usually established by demonstrating the trophozoite or cyst form of *Giardia lamblia* in the stool. Approximately 70 to 97% of infections can be diagnosed with three stool specimens collected 7 days apart.[15,16] When suspicion is high, but a diagnosis cannot be established by direct stool examination, an attempt can be made to identify the organism in the duodenum. The enterotest capsule or a duodenal aspirate can be used to recover the organism. The material should be examined both as a wet mount and as a permanent stain.

Several tests have been developed to identify *Giardia* antigens in the stool using several different techniques, including enzyme-linked immunosorbant assay or monoclonal antibody-based fluorescent techniques. A commercially available kit uses an immunochromatographic strip-based detection for *Entamoeba histolytica, Giardia lamblia,* and *Cryptosporidium parvum.* This test requires the use of fresh or frozen stool, since formalin interferes with the *Entamoeba* reagents. Another commercially available kit tests for *Entamoeba histolytica* and *Cryptosporidium parvum;* this kit can be used with formalin-based stool preservatives as well as fresh or frozen stool. Many of these newer methods are being used to screen patients suspected of having giardiasis or those who may be involved in an outbreak situation.[17,18]

Detection of anti-*Giardia* antibodies in the serum is currently not available for the detection of giardiasis.

Treatment

If giardiasis is diagnosed, the patient should be treated even if asymptomatic. In the majority of cases, metronidazole is the drug of choice. Alternative drugs include quinacrine and furazolidone. Consideration should be

given to treatment of sexual contacts of confirmed cases as well as to individuals exposed during epidemics.

SUMMARY

Sexual activity can be a method of transmission for several important parasitic diseases, including amebiasis and giardiasis. Oral-anal and oral-genital contact predispose male homosexuals to infections with these enteric pathogens. Both of these organisms may cause acute and chronic illnesses as well as other abdominal symptoms. Most gay men with amebiasis are asymptomatic. Both diseases can frequently be diagnosed on routine stool examination, and multiple treatment regimens exist for both.

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